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Anesthetic Management of the Chemically Dependent Patient

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Americans spend over $200 billion annually for self-medication by nonprescription drugs to alter their perception of reality: ethanol, caffeine, nicotine, legal nonprescription nostrums, and illegal street drugs.

The societal demand for chemical substances of abuse has reached epidemic proportions in our population. There is some beginning evidence that its incidence and prevalence has peaked, but major problems remain. This phenomenon has several characteristics: (1) the infiltration of chemical abuse into all strata of society regardless of race, creed, education, or socioeconomic status; (2) the persistence of chemical abuse in spite of intensive media, medical, and governmental efforts to discourage drug abuse; and (3) the tendency to polydrug abuse (the use of several chemicals simultaneously with substantially different pharmacological properties).

At least 10% of the American population above age 14 exhibit substantial health problems associated with ethanol (ethyl alcohol) abuse. Minimally, there are 500,000 heroin addicts in the United States. At least 22 million Americans admit to at least one use of cocaine, and 5 million use cocaine regularly.[1] The number of persons abusing hallucinogens (lysergic acid diethylamide [LSD], phencyclidine, and congeners), central nervous system (CNS) depressants (barbiturates and congeners, benzodiazepines), marijuana, amphetamines, hydrocarbon inhalants, and "designer" street drugs is unknown but surely substantial.

Based upon these epidemiological estimates, the dental practitioner may minimally anticipate that one in five or six dental patients (35 million out of 200 million) is actually or potentially impaired psychologically and/or physically by chemical substance abuse, which may adversely affect their response to dental treatment.

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RECOGNITION OF CHEMICAL SUBSTANCE ABUSE

The first step in the safe management of the chemical substance abuse patient is recognition that the problem exists. It is highly unlikely that any health history form will detect the current drug abuser, as denial is at the center of the problem. The exception is the recovering drug abuser who may admit the past and specifically request that mind-altering drugs not be employed.

Therefore the identification of the chemical substance abuser prior to dental treatment by psychological or physical profile is of paramount importance. Such recognition may be relatively easy, very difficult, or essentially impossible. The patient with chronic alcoholic deterioration (facial signs, tremors, belligerent and defiant behavior, jaundice) may be easy to identify, but the beginning cocaine abuser may readily escape detection. General neglect of personal health, including dental care,[2] may be the hallmark of the drug abuser well on the way to death from the disease but is not the general rule.[3,4] Some useful clues of substance abuse may include a greater prevalence of dental caries (particularly on buccal surfaces) and periodontal disease, oral carcinoma in young individuals related to marijuana and alcohol abuse, bruxism, and xerostomia.[2] Other signs may be a reduced tolerance to pain, manipulative behavior to attain opioid analgesics, a history of hepatitis or AIDS, an inordinate fear of needles, febrile illness (low grade septicemia from intravenous injections), and deteriorated appearance.[3,4]

To aid in the detection of the chemical substance abuser and allow for the safe anesthetic management and subsequent treatment of such a patient, it is necessary to understand the major psychological and physical manifestations of the various forms of drug abuse.

PATHOLOGY OF SUBSTANCE ABUSE

The harmful effects of substance abuse may be relatively specific for the particular drug involved or more generally shared among several types of chemical abuse. Also the signs and symptoms may be of an acute or chronic nature.

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Specific Pathology of Abused Substances

Ethanol. No other drug currently available and taken to excess has as many deleterious effects as ethyl alcohol. The major systems affected are the CNS, gastrointestinal tract, cardiovascular and musculoskeletal systems, skin, blood, and endocrine and metabolic regulatory systems.

CNS pathology includes anxiety, depression, aggression, antisocial behavior, cortical and cerebellar degeneration, paresthesias, encephalopathy, Wernicke's disease (nystagmus, ataxia, paresis) and Korsakoff's psychosis (disorientation, disordered memory). Esophagitis, esophageal varicosities, gastritis and ulcer formation, pancreatitis, and liver disorders (jaundice, fatty liver, hepatitis, cirrhosis) are all gastrointestinal manifestations of ethanol toxicity. Cardiovascular difficulties include cardiomyopathy, depressed left ventricular function, hypertension, increased triglyceride levels, conduction defects (altered T waves and PR intervals, bundle branch block), and rhythm disturbances (atrial and ventricular premature beats, tachyarrhythmias, and fibrillation).[5] Muscle myopathy and osteoporosis may occur. Skin symptoms include rhinophyma (red, beefy nose), pruritis, and telangiectasis (spider angiomas). Disorders of the hematologic system are seen as macrocytic anemia, decreased leukocyte chemotaxis, thrombocytopenia, and reduced prothrombin synthesis. Endocrine and metabolic difficulties include hypoglycemia, decreased serum albumin and total protein, reduced testosterone synthesis, and gynecomastia.

Opioids. Acute intoxication with opioids (heroin, morphine, codeine, meperidine, methadone, pentazocine, propoxyphene) is characterized by decreased blood pressure, respiration, and body temperature along with pupillary constriction (possible dilation with meperidine or extreme hypoxia), depressed reflexes, stupor, and coma.[6] Seizures may be seen with pentazocine, propoxyphene, or meperidine.[7]

Complications with subcutaneous heroin ("skin popper" amyloidosis) include skin infections, diabetes insipidus, and the nephrotic syndrome with renal insufficiency.[8] Parenteral pentazocine abuse is manifested by fibrous ("woody") myopathy and deep skin ulcerations with hyperpigmentation and muscle exposure.[9] Cardiac pathology, namely cardiomyopathy and acquired or congenital valvular abnormalities, is prominent in opioid addicts.[8] At autopsy, 48% of opioid abusers with cardiac disease have healed or active infective endocarditis.[10]

Central Nervous System Depressants. The signs and symptoms of CNS depressant overdosage (barbiturates, benzodiazepines, chloral hydrate, glutethimide, methaqualone) include respiratory and cardiovascular depression, confusion, delirium, depressed tendon reflexes, ataxia, slurred speech, nystagmus, and cardiac arrhythmias, the last particularly with chloral hydrate.[6]

Amphetamines. Acute CNS stimulant intoxication (amphetamines, methylphenidate, phenmetrazine, phenylpropanolamine) is manifested by depressed respiration, increased heart rate and body temperature, delirium, hallucinations, dilated and reactive pupils, hyperactive tendon reflexes, agitation, tremors, sweating, and convulsions.[6] Amphetamine abusers are prone to intracranial hemorrhage, ischemic stroke, carotid stenosis and occlusion, cardiomyopathy, and acute pulmonary edema. Smokable methamphetamine ("LA Ice," "Shabu") may induce prolonged acute psychosis, extreme paranoia, hallucinations, and destructive behavior.

Marijuana. Acute cannabis intoxication (marijuana, hashish, THC, hash oil, sinsemilla) may be manifest as the acute brain syndrome (acute panic, delusions, aggression, disorientation, psychosis).[11] More commonly, chronic marijuana use may elicit tachycardia, conjunctivitis, motor incoordination, inflamed uvula (hashish), altered time perception, euphoria, memory impairment, increased appetite, xerostomia, decreased reaction to sensory stimuli, and the antimotivational syndrome (passivity, apathy, moodiness, decreased concentration, slovenly appearance).[6,11]

Hydrocarbon Solvents. Inhalation of various hydrocarbons (toluene, butane, trichlorethylene, trichlorethane, chlorofluorocarbons) found in gasoline, paint and thinners, lighter fluid, antifreeze, dry cleaning fluids, cements, and nail and hair lacquers results in CNS, liver, renal, neurologic, and cardiovascular toxicity. Signs and symptoms of solvent inebriation include euphoria, ataxia, dizziness, irritability, abdominal pain, and delusions.[7,12] Oropharyngeal manifestations include eczema around the mouth, tracheitis, and bronchitis.[12] Excessive lacrimal and nasal secretions are common. Neurologic symptoms include tremors, myoclonic seizures, and exaggerated jaw-jerk reflexes.[13] Chronic use may result in renal tubular and liver necrosis. Sudden, unexpected death may be due to anoxia, respiratory depression, vagal inhibition, or cardiac arrhythmias possibly due to inhalant-induced myocardial sensitization to catecholamines released from the adrenal medulla by fear, carbon dioxide accumulation, or rapid movement (running).[12]

Anticholinergics. Drugs with cholinergic blocking properties (atropine, scopolamine, propantheline, benztropine, trihexyphenidyl, henbane, jimson weed) induce thirst, blurred vision, dehydration, flushing, dry skin and mucous membranes, dilated fixed pupils, dis-

torted time perception, reduced memory, increased body temperature and heart rate, amnesia, visual hallucinations, excitement, violent behavior, and respiratory failure.[6,14]

Hallucinogens. LSD and other hallucinogens, such as mescaline, psilocybin, STP (2,5-dimethoxy-4-methylamphetamine), DMT (N,N-dimethyltryptamine), and MDMA (3,4-methylenedioxymethamphetamine), induce increased heart rate and blood pressure, hyperreactive tendon reflexes, increased body temperature, euphoria, facial flushing, anxiety, panic, paranoia, time and visual distortions, and visual hallucinations.[6] Additionally, phencyclidine (Angel Dust) may cause extreme hyperactivity, mute behavior, nystagmus, amnesia, muscle rigidity, and impulsive, violent behavior.[6]

Anabolic Androgenic Steroids. Chronic use of the androgenic steroids may result in cholestatic hepatitis, hepatic cysts, hepatomas, testicular atrophy, gynecomastia, and reduced high density lipoproteins in males and menstrual irregularities, alopecia, deepened voice, and facial hair in females.[15] Fluid retention, hypertension, and thickened skin particularly on the back occur in both sexes.[15] Anabolic steroid use is associated with manic episodes, mental depression, delusions, hallucinations, irritability, grandiosity, and violence possibly resulting in homicide.[16]

Cocaine. The CNS effects of cocaine are manifested as euphoria, anxiety, paranoia, increased motor activity, repetitive behavior, tremors, hyperthermia, sleep and eating disturbances, visual, auditory, or tactile hallucinations (insects crawling on the skin), and convulsions.[5,17] Other pathology includes nasal stuffiness and rhinitis, pulmonary edema, pneumomediastinum, intestinal ischemia, renal infarction, necrosis of the nasal septum, and black sputum.[5,17] Neurological toxicity includes agitation, tonic-clonic seizures, headache, focal neurologic deficits, and transient loss of consciousness.[18] Cerebrovascular effects include ischemic stroke, transient ischemic attacks (TIAs), vasculitis, and intracerebral and subarachnoid hemorrhage.[18] The cardiac complications of cocaine are seen as myocardial ischemia and infarction, coronary artery spasm, and arrhythmias.[5,17,18]

Generalized Pathology of Substance Abuse

Three types of generalized tissue pathology are common to several forms of chemical substance abuse: pulmonary, skeletal muscle, and cardiac/cerebrovascular. Each may constitute a major medical emergency and may result in patient fatality.

Pulmonary. Substance abuse toxicity in the respiratory tract can be classified as (1) irritational (inflammatory-allergic), (2) infectious, and (3) barotraumatic. The signs and symptoms of irritational trauma are cough, chronic and acute bronchitis, asthmatic bronchospasm, dyspnea, pulmonary edema, black sputum (from the tarry residues of butane torches used to heat cocaine), foreign body granulomatosis (pulmonary vascular reactions to filler substances such as talc and cellulose), and hemorrhagic vasculitis from hydrocarbon inhalation.[19–21]

Infectious complications include pneumonia, lung abscess, pulmonary artery aneurysm, tuberculosis, and septic pulmonary embolization.[19] The common practice of breath holding and the Valsalva maneuver during inhalation of cocaine, marijuana, and hydrocarbons results in subcutaneous emphysema, mediastinal crepitus (Hamman's sign), pneumothorax, pneumomediastinum, and pneumopericardium.[20,21]

Skeletal Muscle. Rhabdomyolysis (skeletal muscle injury and subsequent lysis) is a sequela to the use of cocaine but is also seen with the abuse of ethanol, heroin, amphetamines, toluene, barbiturates, and phencyclidine.[22] The signs and symptoms are thoracic pain (mimicking myocardial ischemia), bloody sputum, flank, leg, and arm pain, intravascular clotting, hepatic dysfunction, and acute renal failure with myoglobinuria (myoglobins and heme in the urine). Drug-induced rhabdomyolysis is characterized by sudden onset in apparently healthy individuals without evidence of prolonged muscle compression, coma, or status epilepticus.[23] Metabolic abnormalities include increased serum creatine phosphokinase (CPK), hyperkalemia, hyperuricemia, hematuria, and myoglobinuria.[23,24] The etiology of drug-induced rhabdomyolysis includes vasoconstriction, hypoxia, seizures, and volume depletion. Fatalities result from respiratory distress, hyperkalemia, acute renal failure, and particularly disseminated intravascular clotting.[23,24]

Cardiac/Cerebrovascular. The use of cocaine by any route of administration (intranasal, inhalation, intravenous) is associated with potentially severe cardiac and cerebrovascular pathology in young individuals (ages 19 to 48). From 1978 to 1990, at least 39 cases of cerebrovascular accident (CVA) were reported in the medical literature related to cocaine ingestion.[25–27] This toxicity may manifest as intracerebral hemorrhage, subarachnoid hemorrhage, or cerebral thrombotic infarction.[26,27] Signs and symptoms include headache, confusion, agitation, aphasia, ataxia, blurred vision, lethargy, and hemiparesis.[27] Symptom onset may be within minutes to hours after cocaine use.[27] These CVAs are less commonly associated with heroin, amphetamines, methylphenidate, and phencyclidine abuse.[28] The etiol-

ogy of these CVAs include cerebral vasospasm, embolic infarction, microvascular occlusion, vasculitis, and arteriovenous malformations.[26–28]

From 1978 to 1988, 42 cases of cardiac pathology due to cocaine abuse were reported in the medical literature[25] as follows: acute myocardial infarction with arrhythmias (33), arrhythmias alone (5), cardiomyopathy (2), myocarditis (1), and myocardial ischemia (1). Five cases of myocardial infarction and two with arrhythmias alone were fatal.[23] There are now over 60 reported cases of myocardial infarction due to cocaine abuse.[29,30] Many of these individuals had no evidence of coronary artery disease.[25] As with the CVAs associated with cocaine, the myocardial pathology began from immediately after cocaine use to several hours later.[25] The etiology of myocardial cocaine toxicity may include catecholamine-induced platelet aggregation, coronary artery spasm, or increased myocardial oxygen demand.[31] Various adulterants in the street drug preparations cannot be excluded as mitigating factors. Contraction band necrosis of myocardial muscle is common in cocaine-associated fatalities.[25]

It is apparent that cocaine use by dental patients from minutes to 6 or more hours before dental treatment may pose a serious risk for acute cardiac or cerebrovascular morbidity or mortality totally unrelated to the dental anesthetic or treatment procedures. Such events may occur in young individuals in whom no such problems could reasonably be anticipated.

DRUG INTERACTIONS

Documentation of harmful drug interactions in dental patients with chemical dependence and abuse problems are virtually nonexistent. Many reports are only anecdotal in nature.

There are little or no data on general anesthetic requirements in sober or acutely intoxicated alcoholics.[32] It is anecdotally reported that general anesthetic induction in alcoholics is difficult and that the minimum alveolar concentration (MAC) may be decreased in acute alcohol intoxication and increased in chronic alcoholism.[33] Chronic alcoholism has no effect on thiopental pharmacokinetics[34,35] but no such studies have been performed in acute alcohol intoxication. Other CNS depressants (opioids, antipsychotic neuroleptics, antihistamines, benzodiazepines, sedative-hypnotics) are likely to be additive with acute alcohol ingestion. Chronic heavy ethanol ingestion may increase the hepatotoxic effect of acetaminophen, and the concomitant ingestion of ethanol and aspirin may increase gastric bleeding. Metronidazole and certain cephalosporins (cefoperazone, cefamandole, cefotetan) may precipitate a disulfiram-like reaction with ethanol ingestion.

Epinephrine in local anesthetic solutions may increase the tachycardia seen with marijuana[36] or have no effect.[37] Marijuana may decrease the MAC of general anesthetics and exhibit CNS depression with opioids and barbiturates.[33]

Little or no information is available on the potential synergism between the cardiac depressant (local anesthetic) or cardiac stimulant (adrenergic) effects of cocaine and local anesthetics with adrenergic vasoconstrictors. Catecholamine excess in pheochromocytoma and cocaine intoxication may induce similar contraction band necrosis of the myocardium. Cardiac cocaine effects may be enhanced by the postjunctional action of catecholamines on the adrenergic receptors.[39] The coronary artery vasoconstrictive effects of cocaine are enhanced by propranolol.[39]

TREATMENT MODIFICATION FOR SAFE ANESTHETIC MANAGEMENT

If possible, the chemically-dependent dental patient must be identified. Drug interactions, although few in number and severity, should be avoided.

For the occasional asymptomatic substance abuser with no physical manifestations of the disease: (1) outpatient general anesthesia should be avoided if possible; (2) medical consultation may be advisable; (3) for conscious sedation, a benzodiazepine and local anesthesia should be considered; (4) intraoperative electrocardiographic, pulse, and blood pressure monitoring is indicated; and (5) the patient should be warned of potentially serious morbidity or mortality (informed consent). For the chronic substance abuser with evidence of mild debilitating systemic pathology, the above conditions apply with emphasis on medical consultation and consideration of hospitalization. For the chronic user with severe manifestations of substance

Table 1. Treatment Modifications for Safe Anesthetic Management

Identification of substance abuser
Avoidance of drug interactions
Occasional substance abuser
Avoid outpatient general anesthesia
Medical consultation if indicated
Benzodiazepines and local anesthesia
Intraoperative monitoring of heart rhythm, pulse, and blood pressure
Informed consent on morbidity and mortality
Chronic abuser with mild systemic effects
All of the above plus medical consultation
Possible hospitalization
Chronic abuser with serious systemic effects
Emergency care only
Hospitalization

abuse, treatment should be restricted to emergency care in the hospital environment.[25] These recommendations are summarized in Table 1.

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